REMARKS

Applicants have amended Claims 1, 3 and 4 for clarity herein and canceled non-elected Claim 15. No new matter is contained in the amendments. Reconsideration of the present application and allowance of pending Claims 1-14 are respectfully requested in view of the amendments and following remarks.

1. Rejections under 35 U.S.C. § 102

Claims 1-14 were rejected under 35 U.S.C. § 102(b) as being anticipated by Alnemri et al. (U.S. Patent No. 5,786173) (hereinafter "Alnemri"). In rejecting Applicant's responses on May 15, 2008, Examiner asserted that Alnemri taught the same physical steps as the claimed method in determining and influencing the amount or activity of caspase-10 or caspases-10 isoforms.

Applicants respectfully traverse the rejection as follows. A person skilled in the pertinent art would recognize the difference between the claimed method which is directed to non-apoptosis pathways, while Alnemri's teaching focus on apoptosis pathways. However, in an effort to advance prosecution, Applicants hereby amend Claims 1, 3 and 4 without prejudice to further clarify the distinction. The amended independent Claim 1 is directed to determining and influencing the amount or activity of caspase-10 or caspase-10 isoforms in a cell or an organism, *which is affected by non-apoptosis signals* emanating from death receptors or *non-apoptosis signals* emanating from non-death receptor members of the TNF receptor family, *regardless of the disease being treated*, as suggested by the Examiner. Claims 3 and 4 are amended to reflect the change. The amendment therefore rendered the rejections moot and Applicants respectfully request the 102(b) rejection be withdrawn.

The present invention, as clarified in the amended claims, is directed to a method of monitoring and modulating a disease-associated activatory process by determining and influencing the amount or activity of caspase-10 or caspase-10 isoforms that is affected by non-apoptosis signals emanating from death receptors or non-apoptosis signals emanating from non-death receptor members of the TNF receptor family, wherein the non-apoptotic signaling is generated regardless of the disease being treated.

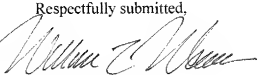
In comparison, Alnemri is directed to the nucleic acid sequence and polypeptide of Mch 4 and Mch 5(caspase 10 isoforms), as well as the making thereof. Although in its specification, Alnemri mentioned use of Mch 4 and Mch 5 as potential means of disease diagnose or treatment, it was expressly limited to programmed cell death (apoptosis) mediated diseases. *See* Alnemri, Column 8, lines 45-48; Cloumn 8, line 65- Column 9, line 3. Therefore, Alnemri does not teach or suggest a method relying on non-apoptosis signaling, as further clarified in the present invention.

CONCLUSION

Applicants believe that the present application, as amended, is now in condition for allowance. Favorable reconsideration of the application as amended is respectfully requested. The foregoing is submitted as a full and complete response to the Final Office Action mailed August 11, 2008.

No fees are believed due at this time. However, please charge any fees that may be due, or credit any overpayment, to Deposit Account 19-5029 (Ref. No.: 18744-0030). In addition, if there are any issues that can be resolved by a telephone conference or an Examiner's amendment, the Examiner is invited and encouraged to call the undersigned attorney at (404) 853-8000.

Respectfully submitted,



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